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Sources and Consumption of Antioxidants in the Diet

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ABSTRACT

Vitamin E is the most important tissue antioxidant in preventing or controlling non-specific reactions from various oxidizing species produced in normal metabolism. Through this action, the vitamin protects polyunsaturated fatty acid loss from phospholipids and consequent membrane damage. The U.S. diet has an abundance of vitamin E and normal individuals accumulate effective amounts in their tissues, which is consistent with the latest recommended dietary allowances for vitamin E as indicated by the National Research Council.

INTRODUCTION

One may reasonably ask why there should be an interest in antioxidants in our food supply. Are they beneficial or harmful? We know from biochemical studies that in the body's tissues there are chemical reactions which under some circumstances could lead to metabolic problems and tissue damage. These reactions produce free radicals or oxidizing species that may react readily with cell components. Such systems as the microsomal mixed function oxidases, the xanthine-xanthine oxidase system, cyclooxygenase and various other enzymes that produce hydrogen peroxide, superoxide or singlet oxygen, all may contribute to potentially damaging conditions in vivo. Fortunately, tissues also contain a varied system of defense against oxidant damage, and primary components of this system are antioxidants.

Foremost is vitamin E, since a deficiency of this vitamin leads to many cellular changes readily explained by its antioxidant action. Other nutrients which also can demonstrate an antioxidant effect, via metal scavenging and under limited conditions, are ascorbic acid, cystine, histidine, tryptophan and intact proteins. Numerous enzymes in tissues also will destroy oxidizing species: catalase, glutathione reductase, glutathione peroxidase (both selenium containing and non-selenium containing), and superoxide dismutase. It should be mentioned that the trace element, selenium, is not an antioxidant, but when incorporated into glutathione peroxidase it readily destroys peroxides.

Two types of antioxidants in the diet will be considered, the natural antioxidant vitamin E, and the synthetic antioxidants added in manufacturing of a multitude of food products. Of the many synthetic antioxidants available, only 2 were approved by the Food and Drug Administration for use in human food in the past. These are BHT (butylated hydroxytoluene) and BHA (butylated hydroxyanisole). These are permitted in fats and oils at a concentration of 0.02%, and also are added to packaging materials. However, BHT is no longer considered acceptable and its use has been stopped in most lipid-containing foods in the U.S. and other countries. In extensive animal testing 20 to 30 years ago, these compounds generally were found to be much less active than vitamin E in preventing the classical signs of vitamin E deficiency. The amounts required in the diet were 20-100 times that of vitamin E, and often bordered on a toxic level. Studies of the metabolism of BHT and BHA in man, using isotopic labeling, revealed that the compounds were rapidly excreted from the body, 80-90% in the urine within 7 days and the remainder in the feces. Furthermore, they are oxidized to 5 or more metabolites. In terms of the amounts that may be ingested daily by man from the U.S. food supply, probably only a few milligrams, it cannot be considered that these two antioxidants make a significant contribution to the body's overall antioxidant defense system. In preventing certain experimentally produced cancers in laboratory animals, these compounds must be in the diet at relatively high levels, 0.5% or more. This would be the equivalent of about 2.5-3 g per day for man.

Vitamin E is the most important dietary component contributing to antioxidant defenses in tissue. Vitamin E is a collective term comprising 8 compounds synthesized by plants. These fall into 2 classes, the tocols having a saturated side chain, and the tocotrienols having an unsaturated side chain. Within each class there are 4 "vitamers," designated alpha, beta, gamma and delta, which vary in the number of methyl groups on the chroman ring. Of these 8 compounds, only 4 have nutritional significance: alpha, beta, and gamma-tocopherols and alpha tocotrienol. When tested in animals for their vitamin E activity, the relative activities are: alpha tocopherol 100, beta tocopherol 30, gamma tocopherol 10, and delta tocopherol 1. In the tocotrienol series the activities are alpha 30, beta 5, and gamma and delta, < 1 (1).

According to their relative abundance in the diet and their relative biological activities, it can be estimated that of the total vitamin E activity in the U.S. food supply 75% comes from alpha tocopherol, 20% from gamma tocopherol, and 5% from beta tocopherol and alpha tocotrienol. Even though gamma tocopherol has only one-tenth the biological activity of alpha tocopherol, it is present in our diet at twice the amount of alpha tocopherol and thus makes a significant contribution. The U.S. diet may be unique in this regard compared with other western countries because of our relatively high consumption of soybean and corn oils, in which gamma tocopherol exceeds alpha tocopherol

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three- to five-fold (2). Once γ-tocopherol is incorporated into cell membranes its antioxidant activity can be as high as 37% that of alpha tocopherol (3). γ-tocopherol is slightly more poorly absorbed than alpha and also exhibits a faster turnover in the tissues.

The richest sources of vitamin E in the U.S. diet are vegetable oils (Table I). In the U.S., soybean oil accounts for about 70% of edible vegetable oils. During processing there are small losses of the tocopherols, but even the hydrogenated shortenings retain relatively high levels. Probably vitamin E is the most widely distributed vitamin in foods. The content in other foods is shown in Table II. Analyses of composite meals by several investigators (4) have shown that U.S. diets contain about 5-13 mg alpha tocopherol equivalent (7.5-19 IU). The range depends primarily on the vegetable fat content of the diet and total calories. Generally, low fat diets will have less vitamin E than high fat diets. Because of the relatively high content of fat in the U.S. diet, and our wide use of vegetable oils, the U.S. food supply is probably the richest in the world in vitamin E, and published values support this (4).

The amount and type of fat in the U.S. food supply has changed markedly since 1950, with a shift away from animal fat to vegetable fat. This change led some nutritionists to question the vitamin E adequacy of our diet, since experiments in animals had shown that increased amounts of polyunsaturated fat increase vitamin E requirements (5). These fears were unfounded, however, because it was not considered that the dietary sources of the polyunsaturated fat, vegetable oils, also are the richest sources of vitamin E. As shown in Table III, from 1950 to 1970, while the amount of oils increased from 21.2 lbs per person per yr to 36.4 lbs, the alpha tocopherol increased from 2.63 g per person per yr to 3.35 g. When the contribution of gamma tocopherol is included, the ratio of mg vitamin E activity:g linoleic acid was 0.61 in 1950 and 0.57 in 1970, not a significant change. Thus, there has not been any deterioration in vitamin E nutrition in the U.S. as a result of changes in type and amount of fat; surveys of human vitamin E status have not shown any change over this period.

One consequence of the change in U.S. dietary fat from animal to vegetable sources has been a marked increase in the intake of gamma tocopherol (Table IV). Analyses of human tissues in 1958 and in 1975 showed an increased content of gamma tocopherol, consistent with the increased dietary content of the gamma "vitamer" over this time period (6). Of interest is the wide variation in the ratio of gamma:alpha tocopherols in different tissues (Table III). It would appear that gamma tocopherol has much more significance for some tissues than for others, but the variation between individuals prevents a generalization.

How do these intakes of tocopherols compare with the Recommended Dietary Allowance for vitamin E as specified by the National Research Council? In the latest recommendations of 1980, the allowance for adult women is 8 mg alpha tocopherol equivalents and for men, 10 mg. These intakes are achieved easily with most diets in this country. It should be noted, however, that low-fat diets generally will have less vitamin E than high-fat diets. At the same time, the need for the vitamin will change as the amount of fat changes, primarily the linoleic acid content.

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TABLE I

Tocopherol Content of U.S. Fats and Oils

Fat	Alpha tocopherol mg/100 g	Gamma tocopherol mg/100 g
Butter	2	0
Lard	1	0
Soybean oil	10-15	75-100
Corn oil	10-20	50-80
Cottonseed oil	40-50	30-40
Safflower oil	25-35	5

TABLE II

Vitamin E Content of Foods as Alpha Tocopherol Equivalents

Food	mg/100 g
Salad oils	15-55
Margarine	10-15
Vegetable shortening	10-15
Peanuts	7
Whole wheat	1
Vegetables	0.1-1
Fruits	0.1-0.3
Meat, fish	0.2-0.5
Eggs	0.5

TABLE III

Change in Dietary Fats and Tocopherols, 1950-1970 (Per person per yr)

	Pounds of fat		Mg alpha-tocopherol	
	1950	1970	1950	1970
Butter	8.7	4.3	119	59
Lard	13.5	7.1	123	64
Soybean oil	9.5	28.5	733	2200
Cottonseed oil	9.5	4.8	1725	872
Corn oil	1.5	2.0	129	173
Peanut oil	0.7	0.7	41	41
Safflower oil	0.0	0.4	0	60
Totals	43.4	47.8	2870	3469

TABLE IV

Change in Tocopherol Intake from U.S. Food Fats, 1950-1970 (Per person per day)

	1950	1970
Gamma tocopherol, mg	11.8	35.5
Alpha tocopherol, mg	7.9	9.5
Total vitamin E activity, (mg alpha tocopherol equivalent)	9.1	13.1